

ticipants in the planning and decisions for health care at the patient, community or national level. It now behooves physicians and the medical profession to spend the time necessary and develop the skills needed to participate fully in this new participatory democracy which has become such an important part of medicine and health care in the society we serve.

—MSMW

## Adult Respiratory Distress Syndrome What To Do Until the Basic Scientist Comes

THE CLINICAL MANIFESTATIONS of diffuse injury to the lung parenchyma vary from fulminant pulmonary edema that may be rapidly fatal to the much more indolent pulmonary fibrosis causing chronic abnormalities of gas exchange, disability and death in some cases. The determinants of the response of the lung to a given injury are unknown and, perhaps more important, the mechanisms by which the injury is delivered are obscure. However, in the past eight to ten years there has been considerable elucidation and definition of the effects of the acute form of lung injury on pulmonary function, and as a consequence of this understanding, more effective approaches to therapy have been developed.

The review of what, for want of a better term, is called the adult respiratory distress syndrome (ARDS) by Petty and Newman in this issue of the *WESTERN JOURNAL* discusses the development of concepts regarding this form of respiratory failure and presents current ideas as to pathogenesis, prevention and treatment. As with other reviews<sup>1,2</sup> of the topic, the present article raises more questions than it answers (an important function of a review article).

One of the more important current questions concerns terminology. The obvious imprecision of the designation ARDS has been discussed previously.<sup>3,4</sup> As noted by Petty and Newman, the lack of general acceptance of this term is apparent by the extraordinary number of synonyms used. Because of a lack of accepted, precisely defined terminology, it is difficult to develop information concerning incidence, natural history and response to therapy. Cooperative studies that at-

tempt to examine any but the most flagrant examples of this form of respiratory insufficiency are doomed to failure because of the inability to define adequately what constitutes the study population. Nevertheless, the catchall term ARDS has been used widely and frequently enough so that it does, in the minds of most physicians, conjure an image of a form of respiratory failure that is quite different from the other common form of respiratory failure—that caused by airway obstruction. For this reason the continued use of the term is of some value, assuming that when specific causes are identified, they will be removed from the ARDS “wastebasket” and treated as separate entities. A good analogy is the clinical and pathophysiologic complex called “heart failure.” A diagnostic evaluation does not end when it is determined that heart failure is present; there is an obligation to find the cause of the heart failure by as complete an examination as is necessary and available. The same philosophy should apply to the patient who is considered to have ARDS.

A whole set of complicated interrelated questions concerning prevention and treatment derive from our lack of knowledge concerning basic mechanisms of injury. It is obvious from reading the “Theories of Pathogenesis” section, and Table 3, in the Petty-Newman paper that many mechanisms alone or in combination are involved. Were we able to identify specific mechanisms in individual patients, then specific therapy could be applied. Theoretic arguments can be raised for the use of corticosteroids and heparin as well as several other agents. However, it has yet to be shown convincingly that any of these compounds has a beneficial effect in patients with ARDS. Here, the problems of imprecise terminology and lack of knowledge of basic mechanisms pose major stumbling blocks. The mechanisms by which acute lung injury occurs were the subject of a recent National Heart, Lung, and Blood Institute workshop that was designed to identify specific research needs.<sup>5</sup> Although the direction provided by this workshop, as well as research currently underway in many centers, certainly holds promise, it appears that we are still several years away from clinically applicable information concerning basic mechanisms. Therefore, for the time being we must be content to treat with supportive rather than curative measures.

We do know that once the lung injury has occurred, the ensuing sequence of events is quite similar regardless of the cause. As Petty and New-

man state, there is considerable evidence indicating that the injury exerts its effect by increasing the permeability of the pulmonary capillary endothelium, thereby allowing fluid to escape initially into the interstitial space and subsequently into alveoli. The increase in pulmonary capillary permeability grossly alters the relationships of the forces that govern fluid movement across the capillary wall. Under these conditions, any increase in the hydrostatic pressure within the capillary causes more fluid to escape from the capillary. Consequently, the fluid management of patients who either have ARDS or are at risk of ARDS developing is of critical importance. Fluids should be carefully administered keeping clear-cut end points in mind, the volume administered being just sufficient to restore or maintain critical organ perfusion with as little increase as possible on the pulmonary capillary (wedge) pressure. (The house staff at San Francisco General Hospital Medical Center have accused us of forcing them to "sneak in at night" to give their patients fluids. We do not deny the accusation.) It should be emphasized that as a guide to fluid replacement in this situation, measurements of central venous pressure are inadequate and frequently misleading. Measurements of cardiac output and pulmonary artery wedge pressure provide much more immediate and accurate information on the need for and effects of fluid administration. However, in many hospitals these are not readily available; therefore, one should rely on other indexes of the state of perfusion including urine output, skin temperature, central nervous system function, blood pH and blood lactate concentrations.

Our current understanding of the pathophysiology of ARDS provides another important approach to prevention and treatment. This is the use of continuous positive pressure ventilation (CPPV). There are more than ample data to document the usefulness of this pattern of mechanical ventilation in patients who have ARDS. Continuous positive pressure ventilation exerts its effect by increasing the volume of gas in partially inflated alveoli or by reinflating collapsed alveoli (or both). Again, by an analysis of the forces acting across the capillary wall it seems logical that early intervention with mechanical ventilation using CPPV may be of some preventive value by lessening the effect of a given injury. By maintaining alveolar inflation, intraalveolar surface tension is reduced. This should lessen the

negativity of the hydrostatic pressure in the pericapillary interstitial space and thereby decrease the pressure gradient between capillary and interstitium. Such a prophylactic effect of CPPV is suggested by two studies showing that in animals ventilated with high tidal volumes the addition of positive end expiratory pressure tends to conserve surfactant and thereby reduces alveolar surface forces<sup>6</sup> and also prevents pulmonary edema.<sup>7</sup>

Although there are still many unanswered questions concerning acute pulmonary injuries, there is at our disposal sufficient information to guide effective supportive care. In addition, there are many situations in which one can anticipate the risks of ARDS and apply the principles of supportive care in a preventive fashion. We await with eager anticipation the revelations that will allow the application of specific curative measures.

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#### REFERENCES

1. Pontoppidan H, Gaffin B, Lowenstein E: Acute respiratory failure in the adult (first of three parts). *N Engl Med* 287: 690-698, 1972
2. Hopewell PC, Murray JF: The adult respiratory distress syndrome. *Ann Rev Med* 27:343-356, 1976
3. Petty TL: The adult respiratory distress syndrome. (Confections of a "lumper.") *Am Rev Respir Dis* 111:713-715, 1975
4. Murray JF: The adult respiratory distress syndrome—May it rest in peace. *Am Rev Respir Dis* 111:716-718, 1975
5. Murray JF, and the staff of the Division of Lung Diseases, National Heart, Lung, and Blood Institutes: Mechanisms of acute respiratory failure. *Am Rev Respir Dis* 115:1071-1078, 1977
6. Webb HH, Tierney DF: Experimental pulmonary edema due to intermittent positive pressure ventilation with high inflation pressures—Protection by positive end-expiratory pressure. *Am Rev Respir Dis* 110:556-565, 1974
7. Wyszogrodski I, Kyei-Aboagye K, Taesch HW Jr, et al: Surfactant inactivation by hyperventilation: Conservation by end-expiratory pressure. *J Appl Physiol* 38:461-466, 1975

## Medicine in Ponape

THE EXPERIENCE of a medical resident in Ponape described elsewhere in this issue serves to draw attention to the sharp differences between medical practice where all the modern resources are available and where they are not. It also emphasizes what it may take to make them available and used appropriately in medically backward areas.

Some similarities between medical practice in the United States and in Ponape stand out with clarity. Patients do not always volunteer all the appropriate information. They may not understand what a physician is asking them or telling them. Cultural attitudes and beliefs may be as